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Berlin Bowel Bothers: Might Adolf Hitler's Gut Problems Have Been Parkinson-Related?

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Abstract
It has been argued that Adolf Hitler (1889–1945) had Parkinson's disease. He also experienced several gastrointestinal symptoms, for which various explanations have been sought, both contemporaneously and by later authors. In this Historical Note, a possible relationship between Hitler's Parkinson's disease and his gastrointestinal symptoms is explored. Specifically, we posit the hypothesis that Hitler may have suffered from small-intestinal bacterial overgrowth (SIBO), thus providing an early example of SIBO occurring as a prodromal Parkinson's disease symptom.

Introduction

A brief history article caught our attention, describing a seemingly insignificant detail about the health of the late German dictator Adolf Hitler (1889–1945) [1]. Among many other ailments – some of which may have been medication-induced – he apparently experienced excessive abdominal gas (meteorism), uncontrollable flatulence, violent bouts of intestinal spasms, constipation, and diarrhoea. Often, attacks of such gastrointestinal complaints would be provoked by psychological stress, meals, or a combination thereof. By his own account, these symptoms had been present since childhood [2]. The diary kept by his personal physician, Theodor Morell (1886–1948), mentions that the first episode of cramping occurred in 1924 [3]. Morell later stated that the complaints had started in 1929 [3]. By any means, as he was appointed personal physician in 1936, the onset of symptoms predated his tenure. Another member of Hitler's medical team, Erwin Giesing (1907–1977), noted that the symptoms became particularly prominent after 1933 [4]. Morell treated Hitler with various drugs, including probiotics and a preparation containing Belladonna-derived atropine and strychnine [2].

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Fig. 1. Hitler enjoying an outdoor meal in Sudetenland during its annexation to Germany, October 3, 1938. Note the abundance of fibre-rich foods such as fruit and bread.

Retrospective explanations given for Hitler's gastrointestinal symptoms include functional bowel disorder [5], irritable bowel syndrome [2, 6, 7], and gastritis/peptic ulcer [8]. Within Hitler's medical team, physicians disagreed on the diagnosis. Giesing considered that the intestinal cramps "may have been of hysterical origin, or may have arisen from an overdose of drugs," [4] Hanskarl von Hasselbach (1903–1981) considered the meteorism to be "probably (. . .) due to a neurosis," [4] while Karl Brandt (1904–1948) presumed the epigastric cramps and vomiting to be the result of "constant strychnin and atropin medication and not of hysteric origin" [4]. Morell, meanwhile, considered Hitler's gastrointestinal symptoms to be caused by abnormal bacterial flora of the intestinal tract, and faecal analysis indeed showed intestinal dysbiosis [9]. Hitler had adopted a vegetarian diet (Fig. 1). Conflicting accounts exist on the timing of, and motivation for, his conversion to vegetarianism. The year 1931 and the suicide of his beloved niece are often mentioned in that regard [3]. Vegetarianism probably aggravated his meteorism [3, 9] as a result of colonic fermentation of plant-based fibres. As evidenced by Morell's diary, this was already recognized at the time [3].

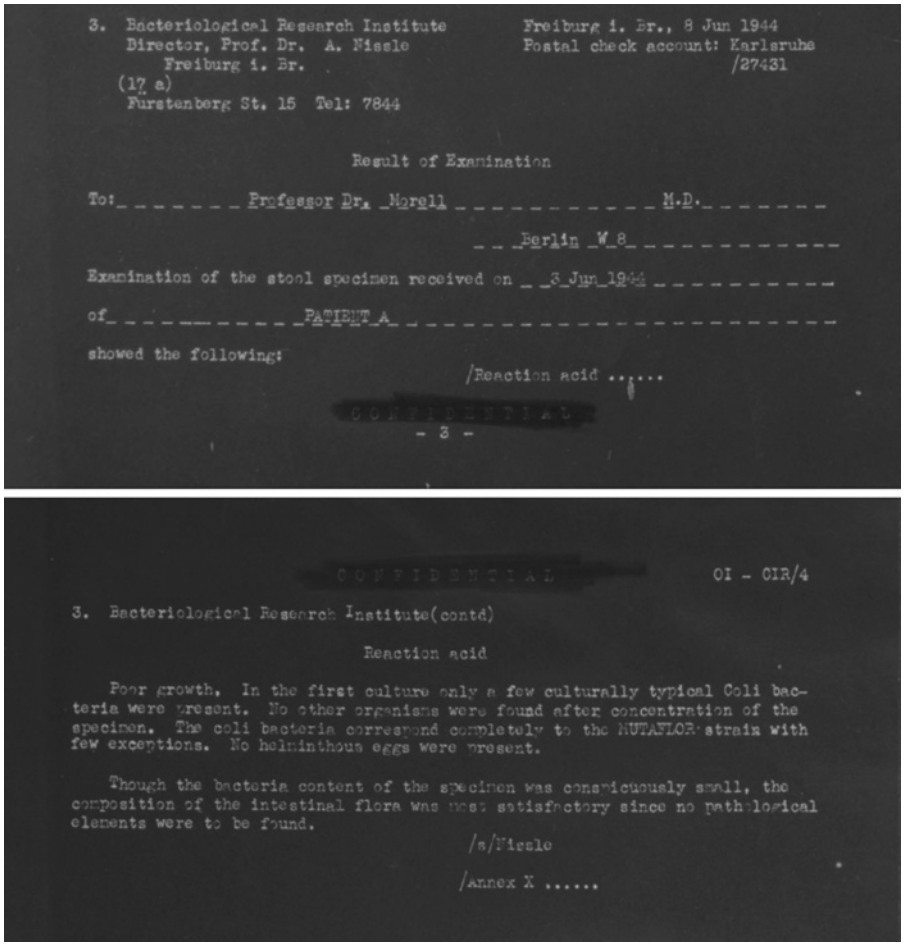
Morell treated Hitler with a preparation that would nowadays be called a probiotic (*Escherichia coli Nissle 1917*), developed by researcher of intestinal flora Alfred Nissle (1874–1965). After 6 months of treatment, Hitler reported that he could once again eat normally without suffering abdominal cramps [3]. Nevertheless, Morell's diary showed that episodes of gastrointestinal symptoms recurred in the following years [3], despite the probiotic treatment continuing (with some interruptions) from 1936 to 1943 [9]. Whereas faecal analysis in 1940 predominantly demonstrated wildtype *E. coli* (perhaps biased by a paucity of bacterial growth in the stool culture, which may also reflect the technical difficulties associated with culturing anaerobes), repeat analysis in 1944 showed all demonstrable *E. coli* bacteria being of the probiotic strain (Fig. 2) [9]. In addition to the probiotic, an apparently prebiotic preparation was intermittently administered, containing ferments that split cellulose, hemicellulose, and carbohydrates [9]. This prebiotic might, paradoxically, have contributed to the meteorism through gas formation resulting from fermentation.

We here propose a new theory/alternative explanation: the possibility that Hitler's gastrointestinal symptoms could have been part of a non-motor prodrome of Parkinson's disease (PD).

Hitler's PD

It has been argued that, starting in his mid-forties or early fifties, Hitler manifested signs consistent with PD. The first visible sign was a left-sided rest tremor, which he used to conceal by keeping his left hand in his pocket or by holding onto an object such as a cane [6]. Documents, photos, and newsreel footage painted a picture of progressive left-sided bradykinesia and tremor, stooped posture, hypomimia, hypophonia as well as micrographia [6, 10–12]. Authors have pinpointed the onset of motor symptoms to either 1933 [13] or 1941 [5].

The possibility of postencephalitic parkinsonism has been suggested as well [5, 12], based upon the supposed occurrence of an encephalitic episode in childhood or early adulthood, as well as reports later in his life of oculogyric crises, dystonic facial spasms, palilalia, autonomic dysfunction, and behavioural abnormalities. However, after analysis of newsreel footage, medical notes, and accounts of contemporaries, neuropsychiatrist Ellen Gibbels found either no reliable evidence of these symptoms or provided alternative explanations for them [5, 14]. In addition, most of those affected by encephalitis lethargica had their acute symptoms between 1918 and 1924 [15], by which time Hitler was well in his adulthood. Several authors have concluded that the documented disease course seems to be most consistent with idiopathic PD [5, 7, 10, 11], given the age of onset, the rate and nature of progression, the presence of various suggestive signs (micrographia, distal tremor, marked asymmetry, several documented non-motor symptoms),



3. Bacteriological Research Institute
Director, Prof. Dr. A. Nissle
Freiburg i. Br.
(17 a)
Fürstenberg St. 15 Tel: 7844

Freiburg i. Br., 8 Jun 1944
Postal check account: Karlsruhe
/27431

Result of Examination

To: Professor Dr. Morell M.D.
Berlin W 8

Examination of the stool specimen received on 3 Jun 1944
of PATIENT A
showed the following:

/Reaction acid ....

CONFIDENTIAL
- 3 -

CONFIDENTIAL OI - CIR/4

3. Bacteriological Research Institute(contd)

Reaction acid

Poor growth. In the first culture only a few culturally typical Coli bacteria were present. No other organisms were found after concentration of the specimen. The coli bacteria correspond completely to the MUTAFLOR-strain with few exceptions. No helminthous eggs were present.

Though the bacteria content of the specimen was conspicuously small, the composition of the intestinal flora was most satisfactory since no pathological elements were to be found.

/s/Nissle

/Annex X .....

Fig. 2. Results of faecal analysis, June 8, 1944. Excerpt from United States Forces, Military Intelligence Service Center OI Consolidated Interrogation Report (CIR) No 4. The few cultured coliform bacteria are shown to be of the Mutaflor strain, the probiotic strain administered to Hitler.

and the lack of definite symptoms associated with postencephalitic parkinsonism.

PD and the Gut

Two phenotypes of PD have been suggested [16]. In the *brain-first phenotype*, alpha-synuclein pathology is thought to start in the nigrostriatal system. In PD patients with a *body-first phenotype*, gastrointestinal changes, including neurodegeneration of the myenteric and submucosal plexus, can occur years before striatal neurodegeneration. Gastrointestinal symptoms such as constipation, caused in part by gastrointestinal dysmotility, have indeed been described to occur as early as 20 years before the onset of motor symptoms. There is now broad consensus that constipation is among the established prodromal symptoms of PD [17]. In the past decade, some research has focused on the occurrence of small-intestinal bacterial overgrowth (SIBO) in PD. This is a state of abnormal bacterial colonisation of the small intestine, which is normally prevented mainly by gastrointestinal motility, but the development of which is facilitated in persons with PD because of their gastrointestinal dysmotility. SIBO is accompanied by symptoms that include bloating, abdominal pain, flatulence, and diarrhoea. It has a potential detrimental effect on both nutrient and medication absorption, the latter resulting in a delayed and decreased effect of levodopa as well as marked response fluctuations, in particular including dose failures and a delayed ON period [18]. SIBO is thought to affect 25–55% of PD patients [19, 20].

We here posit the hypothesis that Hitler could have suffered from SIBO as a prodromal PD symptom. Admittedly, this currently is a speculative possibility, based upon this single historical case. However, although the limited available research has thus far focused on SIBO in patients with established PD, there are various arguments which make it plausible that PD-related SIBO could manifest itself prior to the onset of motor symptoms.

Firstly, one of the most important factors driving SIBO in PD is thought to be gastrointestinal dysmotility, and because this can develop early in the disease course, it might well be possible for SIBO to develop in the prodromal phase, prior to the emergence of any motor manifestation. *Helicobacter pylori* infection, another predisposing factor for SIBO in PD, can precede diagnosis of PD by multiple decades [21]. Inflammatory patterns in blood associated with SIBO have been found in persons deemed to be in a "premonitory state" of PD [22]. Secondly, a role for SIBO in the aetiological pathophysiology of PD has been hypothesised [21]. An altered gut microbiome might be a risk factor for a subsequent development of PD in susceptible individuals [23]; this intestinal dysbiosis – if it exists as an early causative factor – would then by definition have to be present before the onset of motor symptoms. According to this concept, the same dysbiosis could also be a risk factor for development of SIBO.

Likelihood of a Relationship between Hitler's Gastrointestinal Symptoms and PD

Regarding a possible relationship between Hitler's gastrointestinal symptoms and his presumed PD, several hypotheses can be considered: (1) the gastrointestinal symptoms were a direct consequence of PD-related gastrointestinal dysmotility; (2) SIBO, partly induced or perpetuated by PD, caused the gastrointestinal symptoms; (3) intestinal dysbiosis (causing or caused by gastrointestinal symptoms, with or without SIBO) predisposed to the development of PD; or (4) the gastrointestinal symptoms and PD were unrelated (and could, for instance, be explained by another cause of bowel complaints that has a high prevalence in the general population, such as irritable bowel syndrome). We will discuss these options here.

Chronology is an important factor to consider in determining whether Hitler's gastrointestinal symptoms and PD could have been related. In his case, the interval between the onset of gastrointestinal symptoms (ranging from his childhood to 1929, depending on the sources used) and the onset of PD motor symptoms (1933 or 1941) could have been as short as 4 years, or as long as five decades. The shortest interval could be easily reconciled with a prodromal PD symptom, whilst the longest interval would make an aetiological association highly improbable.

The symptoms, the established dysbiosis, and the (at least partial) symptom improvement after probiotic treatment could be regarded as arguments for the presence of SIBO, with or without relation to PD. Furthermore, examination of Hitler's skull fragments showed dental changes possibly related to gastric disease, such as peptic ulcer [8]. Presence of a gastric ulcer might, in addition to providing a partial explanation for Hitler's symptoms such as postprandial epigastric pain and bloating, also serve as a clue for SIBO. *H. pylori*, now known as an important causative agent in peptic ulcers, is highly prevalent in PD patients and can enable the development or exacerbation of SIBO [19, 21]. In addition, diurnal fluctuations in PD motor symptoms may have been present in Hitler, with neurologist Abraham Lieberman describing different performance levels throughout a single day in 1934 [13]. These are, however, unlikely to be malabsorption-related: while Hitler did use a (Belladonna-derived) anticholinergic, which might to some extent have relieved his parkinsonian symptoms and the effect of which would have been dependent on intestinal absorption, his use of this drug seemingly started in 1936, 2 years after the shooting of the film analysed by Lieberman [5].

Unfortunately, details of Hitler's initial faecal examination are not available, precluding a comparison with the microbiome composition described in PD patients' faeces.

Our hypothesis that SIBO was indeed a prodromal feature of PD would be strengthened if Hitler also manifested other prodromal non-motor features of PD. Unfortunately, there is too little information available in the historical literature to speculate about the presence of any other non-motor prodromal symptoms of PD in Hitler. To our knowledge, there are no reports of Hitler complaining about a loss of smell, and on three occasions a normal sense of smell was reported in descriptions of cranial nerve examinations [4, 9]. However, a standard cranial nerve examination is not sensitive to subtle hyposmia, and indeed, specific testing for smell is often left out of standard neurological examinations. Whilst Hitler reportedly had trouble sleeping [12], there is insufficient information available to classify his night-time troubles as a REM sleep behaviour disorder. And while numerous psychiatric diagnoses have been applied retrospectively to Hitler, depression does not feature prominently amongst them.

Finally, the presence of additional abdominal symptoms in Hitler (such as two documented episodes of jaundice [3, 4], the second of which was accompanied by brown discoloration of urine [3], with bilirubinuria demonstrable in between episodes [3]) could indicate an underlying primary gastroenterological disease and serve as an argument against a PD-related aetiology.

As a concluding remark, we obviously acknowledge that diagnosing an individual based on historical accounts and archive footage is an undertaking fraught with

hazards. Indeed, hypothesising on a connection between the different ailments is even more speculative. Armbruster and Theiss-Abendroth [24] highlighted the pitfalls associated with retrospective ("ex-post") diagnosis of Hitler. The paucity of objective, contemporary medical information is just one of the difficulties. Another is the retrospective application of a diagnosis that did not exist during the time, making it difficult to apply diagnostic criteria. Indeed, they demonstrated how a speculative account of a detail in Hitler's medical history (such as this very paper) can unintendedly be referenced in further works as if it were a factual source, building a chain of evidence that is ultimately based upon conjecture. Even so, the dictator's bowel bothers might provide a glimpse of insight into the role and timing of SIBO in PD. This is a topic that is gaining rapid recognition in the Parkinson field as a hitherto underestimated factor that may contribute to complex response fluctuations in levodopa therapy. Further research into this area is warranted.

Statement of Ethics

Ethical approval was not required as this study was based on publicly available data.

Conflict of Interest Statement

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Author Contributions

M.B.: investigation, writing – original draft, and visualization. P.K. and G.W.: writing – review and editing. B.B.: conceptualization, writing – review and editing, and supervision.

Data Availability Statement

Data sharing is not applicable to this article as no new data were created or analysed in this study. Further enquiries can be directed to the corresponding author.

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